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Correlation of Electrophysiological Properties and Hearing Preservation in Cochlear Implant Patients

Dalbert, Adrian ; Sim, Jae Hoon ; Gerig, Rahel ; Pfiffner, Flurin ; Roosli, Christof ; Huber, Alexander

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Correlation of Electrophysiological Properties and Hearing Preservation in Cochlear Implant Patients

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Objective: To monitor changes in cochlear function during cochlear implantation using electrocochleography (ECoG) and to correlate changes to postoperative hearing preservation.

Methods: ECoG responses to acoustic stimuli of 250, 500, and 1000 Hz were recorded during cochlear implantation. The recording electrode was placed on the promontory and stabilized to fix the position during cochlear implantation. Baseline recordings were obtained after completion of the posterior tympanotomy. Changes of the ongoing ECoG response at suprathreshold intensities were analyzed after full insertion of the cochlear implant electrode array. Audiometric tests were conducted before and 4 weeks after surgery and correlated with electrophysiological findings.

Results: Ninety-five percent (18/19) of cochlear implant subjects had measurable ECoG responses. Under unchanged conditions, recordings showed a high repeatability without significant

differences between 2 recordings ($p \leq 0.01$). Ninety-four percent (17/18) of subjects showed no relevant changes in ECoG recordings after insertion of the cochlear implant electrode array. One subject showed decreases in responses at all frequencies indicative of cochlear trauma. This was associated with a complete hearing loss 4 weeks after surgery compared with mean presurgical low-frequency hearing of 78 dB HL.

Conclusion: Extracochlear ECoG is a reliable tool to assess cochlear function during cochlear implantation. Moderate threshold shifts could be caused by postoperative mechanisms or minor cochlear trauma. Detectable changes in extracochlear ECoG recordings, indicating gross cochlear trauma, are probably predictive of complete loss of residual acoustic hearing. **Key Words:** Cochlear implantation—Cochlear implant—Electrocochleography—Hearing preservation—Monitoring—Residual hearing. *Otol Neurotol* 36:1172–1180, 2015.

Intracochlear trauma should be minimized during modern-day cochlear implantation surgeries (1). It has been shown that better performance of cochlear implant recipients with minimal residual hearing is correlated with minimizing cochlear trauma (2–4). In recipients with substantial residual acoustic hearing, it is essential that trauma be minimized for hearing preservation. However, it remains unclear whether with modern electrode designs and surgical techniques, the main reason for postoperative hearing loss is acute intracochlear trauma. Other mechanisms have been proposed, and recent publications have suggested postoperative mechanisms play a relevant role (5,6).

Electrocochleography (ECoG) as a method to assess cochlear function has been known for many years (7). By ECoG, responses of the cochlea and the cochlear nerve to

acoustic stimuli can be recorded. Responses represent remaining cochlear function, which is the basis for residual hearing. Four different potentials contribute to the ECoG signal: cochlear microphonic (CM), summing potential (SP), auditory nerve neurophonic (ANN), and compound action potential (CAP). The ongoing portion of the ECoG signal, the portion of the ECoG signal, which lasts for the duration of the acoustic stimulus, contains the CM, SP, and ANN. The CM is mainly generated by outer hair cells. It is a transducer current in the stereocilia and follows the waveform of the acoustic stimulus. The SP represents a sustained depolarization in the soma of the inner hair cells. The ANN is a neural response and represents the correlate to neural phase-locking. The CAP is not part of the ongoing ECoG response. It is produced by synchronized action potentials across nerve fibers at the onset or offset of sounds.

Several studies that aimed to identify electrophysiological markers of cochlear trauma during electrode insertion in an animal model have been published (8–12). The most consistent and sensitive marker for intracochlear trauma is a reduction in the ongoing ECoG response to a suprathreshold stimulus. This reduction in the

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ongoing response is a more reliable marker than threshold changes or changes of the CAP.

The recording of ECoG during cochlear implantation in humans has been described (5,13–18). It has been shown that the ECoG magnitude is a strong predictor for cochlear implant performance in postlingually deafened adults and children (15,17,18). To our knowledge, 2 reports have been published correlating intraoperative ECoG with hearing preservation after surgery. Radeloff et al. looked at the visual detection threshold of CM during cochlear implantation and found no changes despite complete hearing loss 1 week after surgery in 2 of 4 subjects with deep insertions (5). They suggested that postoperative mechanisms play a major role in hearing loss after cochlear implantation. Mandalà et al. analyzed changes in amplitude and latency of the CAP during several stages of cochlear implant surgery (14). They found that a decrease of CAP amplitude was associated with higher degrees of hearing loss 4 weeks after surgery.

This study aimed the following: 1) to assess ECoG immediately before and after cochlear implantation, 2) to use changes in the ongoing ECoG response at suprathreshold intensities as a marker for changes of cochlear function during cochlear implantation, and 3) to assess the correlation of changes in cochlear function during surgery and hearing preservation 4 weeks after surgery. The expectations were that ECoG responses can be recorded in a majority of cochlear implant recipients with residual hearing and that an unknown fraction of postoperative threshold shifts would correlate with deteriorations of the ongoing ECoG response during surgery.

MATERIALS AND METHODS

The study protocol was approved by the Ethical Committee of Zurich (KEK-ZH-Nr. 2013–0317). It was written in concordance with the Helsinki Declaration. Inclusion criteria were as follows: 1) indication for cochlear implantation after presurgical evaluations at the University Hospital of Zurich, Switzerland; 2) ≥ 18 years old; and 3) residual hearing based on the preoperative pure-tone audiogram. Subjects were recruited and operated on between November 2013 and August 2014. All subjects provided written informed consent before their surgery.

Three different cochlear implant devices have been used in this study: The Cochlear Nucleus CI24RE(CA) with a precurved electrode design for perimodiolar placement, the Cochlear Nucleus CI422 with a slim straight electrode array for lateral wall positioning, and the HiRes90K Advantage cochlear implant with the precurved HiFocus Mid-Scala electrode array designed to achieve a position in the middle of the scala tympani.

Audiometric Assessment

The audiometric assessment was performed in accordance with ISO 8253–1. Pure-tone audiograms were conducted within 6 weeks before surgery and 4 weeks after surgery. Maximum audiometer output was 100 dB HL at 250 Hz and 120 dB HL at 500 and 1000 Hz. Any response reported as vibrotactile or questionably vibrotactile was considered as no response. To represent the remaining mean low frequency hearing, the average of hearing thresholds at 250, 500, and 1000 Hz was calculated. If frequencies with no responses were present at the

maximum output of the audiometer, then the maximum output plus 5 dB was entered to avoid losing data. This approach was used previously by Balkany et al. and Kiefer et al. (19,20).

Percentage of preserved residual hearing (S) and hearing preservation category were assessed according to the recently published hearing preservation classification system by the HEARING group (21), that is, $S = 1 - ([\text{postsurgical PTA} - \text{presurgical PTA}] / [\text{maximum PTA} - \text{presurgical PTA}]) * 100$ (%). PTA is calculated from the pure tone average at the following frequencies: 125, 250, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000, and 8000 Hz. Maximum PTA is the average of the limits of the audiometer. Four hearing preservation categories are defined by the percentage of preserved residual hearing: 1) complete hearing preservation ($>75\%$ of residual hearing preserved), 2) partial hearing preservation ($>25\%$ – 75% of residual hearing preserved), 3) minimal hearing preservation ($>0\%$ – 25% of residual hearing preserved), and 4) loss of hearing (no measurable hearing).

Differences between presurgical and postsurgical hearing thresholds have been determined from the mean low frequency hearing as defined previously. To control for natural progression of hearing loss independent from cochlear implantation, the difference in mean low frequency hearing thresholds as defined previously were assessed in the contralateral ear.

Surgery and ECoG Recordings

All surgeries were performed at the University Hospital of Zurich. A single dose of ceftriaxone 2 g and methylprednisolone 250 mg was provided intravenously at induction of anesthesia. Facial monitoring was used in all subjects. A standard retroauricular incision and an anterior mastoidectomy were performed. The dura of the middle cranial fossa and the digastric ridge were skeletonized, and the posterior canal wall was thinned as much as possible. To open the facial recess, the facial nerve was identified at the stylomastoid foramen and skeletonized. The space between the facial nerve and the chorda tympani nerve was maximally opened. When the round window niche was overhanging, it was carefully reduced with a drill. If the round window was obstructed by a secondary membrane, this membrane was removed for maximal visualization of the round window. After complete visualization of the round window, the recording electrode (Neurosign; Magstim Co., Wales, UK) was placed through the posterior tympanotomy. It was placed on the promontory and left in an unchanged position for the rest of the surgery. Fixation of the recording electrode was achieved by bone wax in the mastoidectomy cavity. If the impedance of the recording electrode exceeded 10 kOhm, then a resorbable gelatin sponge (Spongostan; Ethicon Inc., Sommerville, MA, USA) was placed around the electrode on the promontory. After completion of these steps, baseline recordings were made. Afterward, an anterior-inferior cochleostomy, or a round window insertion, according to soft surgery principles, was performed. The cochlear implant electrode array was slowly inserted, and the insertion site was sealed with periosteum. Postinsertional ECoG recordings were then performed. After completion of the ECoG recordings, the recording electrode was removed, and the wound was closed in layers. Neuroresponse telemetry was performed to confirm function of the implant. The position of the cochlear implant electrode array was assessed using a cochlear view x-ray or digital volume tomography within 4 weeks after surgery.

Recording electrodes were placed on the promontory as described previously (“positive”), in the contralateral preauricular region (“negative”), and on the forehead (“ground”). During

recording, impedance measurements were less than 10 kOhm on all electrodes.

The Navigator Pro stimulation/recording device from Biologic Systems (Mundelein, IL, USA) and the associated AEP software were used for acoustic stimulation and recording. Acoustic stimuli were delivered by sterilized foam insert earphones placed in the ear canal before surgery. Responses to 400 tone bursts with alternating starting phases at 250, 500, and 1000 Hz were recorded. Rise/fall times were 2 cycles shaped by a Blackman window. The plateau phase was 4 cycles at 250 Hz, 10 cycles at 500 Hz, and 20 cycles at 1000 Hz. The stimulus rate was 23.3 kHz. Sound pressure was between 80 and 85 dB HL at 250 Hz, 85 and 95 dB HL at 500 Hz, and 90 and 100 dB HL at 1000 Hz. The recording window was 32 ms, starting 4 ms before stimulus presentation. The sampling rate was 8 kHz for 250 and 500 Hz stimuli and 16 kHz for 1 kHz stimuli. High pass filters were set at 10 Hz and low pass filters at 5000 Hz. For artifact rejection, 47.5 uV was selected.

Repeatability of ECoG recordings under unchanged conditions was assessed by repeated acoustic stimuli of 500 Hz at 85 dB HL or 95 dB HL. Time difference between these 2 recordings was at least 1 minute.

Recordings with disconnected loud speakers to assess the noise level and to control for electrical artifacts were conducted at the end of each session. Sound pressure in the ear canal was monitored by a probe microphone (ER-7C; Etymotic Inc., Elk Grove Village, IL, USA) placed near the tympanic membrane during all recordings.

Data Analysis

The data from rarefaction and condensation phases were stored separately. A difference curve was obtained, subtracting the condensation from the rarefaction phase, and an alternating curve was obtained from the average. Data were exported from the AEP software using AEP to ASCII software from Biologic (Mundelein, IL, USA). MATLAB (MathWorks Inc., Natick, MA, USA) and GraphPad Prism V5.04 (GraphPad Software Inc., La Jolla, CA, USA) were used for further postprocessing.

The spectrum of each response was obtained by fast Fourier transform. Therefore, a time window that isolated the ongoing portion of the ECoG response from the CAP was defined. The

energy content was measured at the frequency of the signal and the frequency of the first harmonic. The sum was defined as the amplitude of the ongoing ECoG response at the frequency of the acoustic stimulus.

Mean noise floor and its standard deviation were determined from all bins within 150 to 200 Hz and 300 to 350 Hz for 250 Hz, within 400 to 450 Hz and 550 to 600 Hz for 500 Hz, and within 900 to 950 Hz and 1050 to 1100 Hz for 1000 Hz. A response was defined as significant if it exceeded the calculated noise floor plus 3 standard deviations.

RESULTS

Intraoperative recordings to acoustic stimuli were obtained in 19 cochlear implant recipients. Subject demographics are summarized in Table 1. Etiology of hearing loss was unknown in 84% (16/19) of subjects. Specific causes for hearing loss were as follows: fetal rubella infection (subject 12), otosclerosis (subject 13), and bacterial meningitis (subject 14). All subjects except subject 14 had no recent changes in hearing threshold before surgery and a history of hearing loss of more than 10 years. Subject 14 had a rapid progression of hearing loss due to meningitis within 3 months. Seven subjects received a Cochlear Nucleus CI422 device, 3 subjects received a Cochlear Nucleus CI24RE(CA), and 9 subjects received a HiRes90K HiFocus Mid-scala.

No complications occurred during surgery. Full electrode insertion could be achieved in all cases. Radiographic controls showed correct positioning of the electrode array and no tip fold-over or kinking.

Postoperative complications occurred in 1 subject. After an uneventful surgery, subject 8 experienced acute vestibular failure on the operated side. The diagnosis was made 1 week after surgery. Treatment consisted of dexamethasone 40 mg for 3 days, followed by dexamethasone 10 mg for another 3 days. Two weeks after surgery, no symptoms persisted, and no corrective saccade could be detected in the head impulse test.

TABLE 1. Subject demographics

Subject No.	Age (yr)	Etiology of Hearing Loss	Duration of Hearing Loss	Side	Round Window Insertion	Cochlear Implant
1	49	Idiopathic	>10 years	L	Yes	Cochlear Nucleus CI422
2	21	Idiopathic	>10 years	R	Yes	Cochlear Nucleus CI422
3	35	Idiopathic	>10 years	L	Yes	HiRes90K HiFocus Mid-scala
4	51	Idiopathic	>10 years	R	Yes	Cochlear Nucleus CI422
5	43	Idiopathic	>10 years	L	Yes	HiRes90K HiFocus Mid-scala
6	52	Idiopathic	>10 years	R	No	Cochlear Nucleus CI24RE(CA)
7	44	Idiopathic	>10 years	R	Yes	Cochlear Nucleus CI422
8	36	Idiopathic	>10 years	R	Yes	HiRes90K HiFocus Mid-scala
9	70	Idiopathic	>10 years	R	Yes	HiRes90K HiFocus Mid-scala
10	62	Idiopathic	>10 years	R	Yes	Cochlear Nucleus CI422
11	21	Idiopathic	>10 years	L	Yes	HiRes90K HiFocus Mid-scala
12	40	Fetal rubella infection	>10 years	L	No	Cochlear Nucleus CI24RE(CA)
13	64	Otosclerosis	>10 years	R	Yes	HiRes90K HiFocus Mid-scala
14	55	Meningitis	3 months	R	No	Cochlear Nucleus CI24RE(CA)
15	76	Idiopathic	>10 years	R	Yes	Cochlear Nucleus CI422
16	58	Idiopathic	>10 years	R	Yes	HiRes90K HiFocus Mid-scala
17	57	Idiopathic	>10 years	R	Yes	HiRes90K HiFocus Mid-scala
18	52	Idiopathic	>10 years	L	Yes	HiRes90K HiFocus Mid-scala
19	65	Idiopathic	>10 years	R	Yes	Cochlear Nucleus CI422

TABLE 2. Audiometric and electrophysiological findings

Subject No.	Presurgical Hearing Thresholds at 250/500/ 1000 Hz (dB)	Mean Presurgical Hearing Threshold at 250, 500, and 1000 Hz (dB)	Postsurgical Hearing Threshold at 250/500/ 1000 Hz (dB)	Mean Postsurgical Hearing Threshold at 250, 500, and 1000 Hz (dB)	Mean Change in Low Frequency Hearing (dB)	Mean Change in Low Frequency Hearing on Contralateral Side (dB)	Hearing Preservation Category*	Percentage of Preserved Residual Hearing* (%)	Difference in ECoG Response at 250 Hz (dB)	Difference in ECoG Response at 500 Hz (dB)	Difference in ECoG Response at 1000 Hz (dB)
1	20/35/65	40	25/45/90	53.3	13.3	5	Complete	77.5	2.3	-4.8	-5.4
2	50/55/70	58.3	60/70/95	75	16.7	-3.3	Partial	70	6.8	-2.8	10.6
3	10/45/105	53.3	15/65/110	63.3	10	-5	Complete	77.4	5.4	2.3	0.8
4	40/50/65	51.7	50/60/115	75	23.3	0	Partial	60.9	-1.9	3.5	0.5
5	40/65/80	61.7	60/95/100	85	23.3	No residual hearing	Partial	52.2	7.4	1.4	8.6
6	75/90/100	88.3	90/100/110	100	11.7	3.3	Partial	55.3	-12.3	2	-0.2
7	55/90/105	83.3	70/105/NR	100	16.7	No residual hearing	Partial	65.8	2.5	1.6	-2.2
8	60/65/75	66.7	65/75/95	78.3	11.7	5	Partial	62.1	7.3	2.4	2.2
9	70/85/NR	93.3	70/90/NR	95	1.7	1.7	Complete	95	0.5	1	NR
10	75/80/80	78.3	NR/NR/NR	—	40	-6.7	Loss of hearing	—	-2.8	-6.4	-25.6
11	75/85/110	90	NR/95/115	105	15	-5	Partial	34.9	-1.1	8.4	NR
12	90/105/115	103.3	NR/NR/NR	—	15	0	Loss of hearing	—	3.7	-1.2	1.9
13	NR/115/115	111.7	NR/110/105	106.7	-5	3.3	Complete	98.1	3.2	-4.9	4.5
14	60/70/85	71.7	NR/110/NR	113.3	41.7	No residual hearing	Minimal	1.8	6.7	7.8	4
15	NR/110/100	105	NR/NR/NR	—	13.3	5	Loss of hearing	—	NR	NR	-2.7
16	65/70/85	73.3	NR/115/115	111.7	38.3	-3.3	Minimal	11.6	-0.3	3.4	-0.7
17	45/90/110	81.7	70/NR/NR	106.7	25	No residual hearing	Partial	48.5	-2.4	NR	NR
18	90/105/115	103.3	NA	NA	NA	NA	NA	NA	3.8	6.5	2.8
19	55/85/115	85	NR/NR/NR	—	33.3	11.7	Loss of hearing	—	NR	NR	NR

NR indicates no response; NA, no data available.

*According to Skarzynski H, van de Heyning P, Agrawal S et al. Towards a consensus on a hearing preservation classification system. *Acta Otolaryngol Suppl* 2013;3-13.

Preservation of Residual Hearing

Audiometric and electrophysiological findings are summarized in Table 2. In subject 18, the postoperative audiometric assessment had to be postponed because of an accident not associated with the cochlear implantation. Therefore, no postoperative hearing thresholds were available.

According to the hearing classification system published by the HEARING group (21), 22% (4/18) of subjects had complete hearing preservation, 44% (8/18) partial hearing preservation, 11% (2/18) minimal hearing preservation, and 22% (4/18) complete loss of hearing 4 weeks after surgery. Mean hearing loss based on the low-frequency average (250, 500, and 1000 Hz) was 19.2 dB (range, -5 to 41.7 dB). Figure 1 compares the mean presurgical and postsurgical hearing thresholds in the low frequencies.

Subjects 5, 7, 14, and 17 had no residual hearing in the contralateral ear. In all other subjects ($n = 14$), mean change in low frequency hearing in the contralateral ear was 0.8 dB (range, -6.7 to 11.7 dB). Only subject 19 showed a threshold shift of greater than 10 dB on the contralateral side.

Electrophysiological Findings

ECoG responses were recordable in 95% of subjects (18/19) (Table 2). In the case with no detectable ECoG

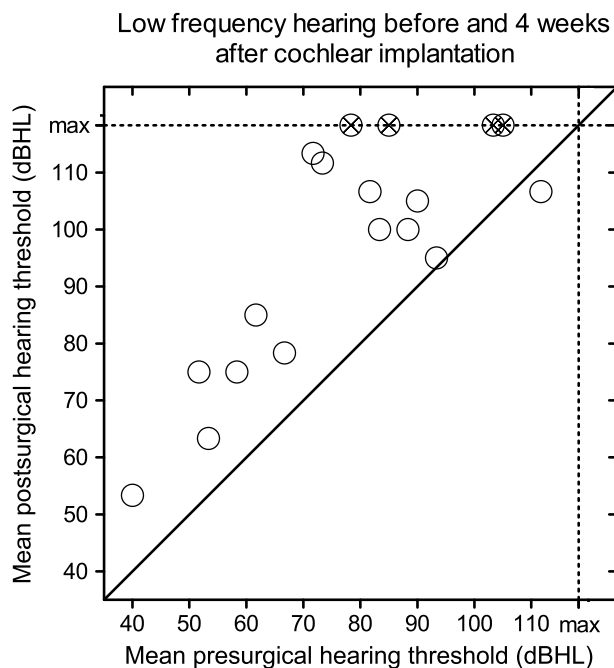


FIG. 1. Comparison of mean low frequency hearing before and 4 weeks after cochlear implantation. Each circle is a subject. The x-axis represents the mean hearing threshold at 250, 500, and 1000 Hz before surgery, the y-axis 4 weeks after surgery. Circles with a cross represent subjects with a complete loss of residual hearing 4 weeks after surgery. Approximately 78% (14/18) of subjects had a low frequency hearing loss of 25 dB or less.

signals (subject 19), a technical problem may have been the reason as the recorded sound pressure in the ear canal was too low (maximum, 56 dB SPL). In all other subjects, the intended sound pressure was reached.

Figure 2 displays 2 examples of ECoG responses before insertion of the cochlear implant electrode array. Neural contribution to the ECoG signal varied between subjects. Indicative for neural contribution is the presence of a CAP and a large contribution to the amplitude of the ongoing signal by the energy at the frequency of the first harmonic. The top row of Figure 2 shows an example where neural contribution is clearly visible, whereas the signal in the bottom row seems to be almost exclusively a hair cell response.

Under unchanged conditions, a high repeatability of ECoG recordings could be detected. It was assessed in 16 subjects. The mean difference between 2 recordings was 0.2 dB with a standard deviation of 2.5 dB. The differences were not significant (Wilcoxon matched-pairs signed rank test, $Z = -0.5688$, $p \leq 0.01$).

Different degrees of change could be detected when ECoG responses before and after insertion of the cochlear implant electrode array were compared. Figure 3 displays 2 examples. The top row shows a case where almost no changes occurred. In the bottom row, a decrease of the amplitude of the ECoG signal could be detected at all 3 frequencies after insertion of the electrode array. Table 2 and Figure 4 summarize the changes in ECoG signal amplitude. Only subject 10 showed a decrease at all 3 frequencies. All other subjects showed no consistent pattern indicating cochlear trauma. Subject 6 showed a larger decrease at 250 Hz. However, at 500 and 1000 Hz, no decrease in response amplitude was detectable. Therefore, 94% (17/18) of subjects showed no clear signs of intracochlear trauma immediately after insertion of the electrode array.

Correlation of Hearing Preservation and Electrophysiological Findings

In subjects 10, 12, 15, and 19, residual hearing was completely lost 4 weeks after surgery. The amount of low frequency hearing loss was 40, 15, 13.3, and 33.3 dB. The complete loss of residual hearing with a low frequency hearing loss of 40 dB corresponded to the case with a decrease of ECoG amplitudes in all 3 recorded frequencies during cochlear implantation (subject 10). In subject 12 with a 15 dB hearing loss, no relevant changes in ECoG signals occurred (3.7 dB at 250 Hz, -1.2 dB at 500 Hz, and 1.9 dB at 1000 Hz). In subject 15 with a 13.3 dB hearing loss, an ECoG response could only be detected at 1000 Hz before insertion. The postinsertional ECoG response showed also no relevant change (-2.7 dB). In subject 19 with a 33.3 dB hearing loss, no ECoG signals could be detected, which was probably due to technical reasons as mentioned previously.

Subjects 14 and 16 showed comparable amounts of low frequency hearing loss with 41.4 and 38.3 dB, despite some remaining acoustic hearing after surgery. Both subjects showed no signs for acute intracochlear trauma in the

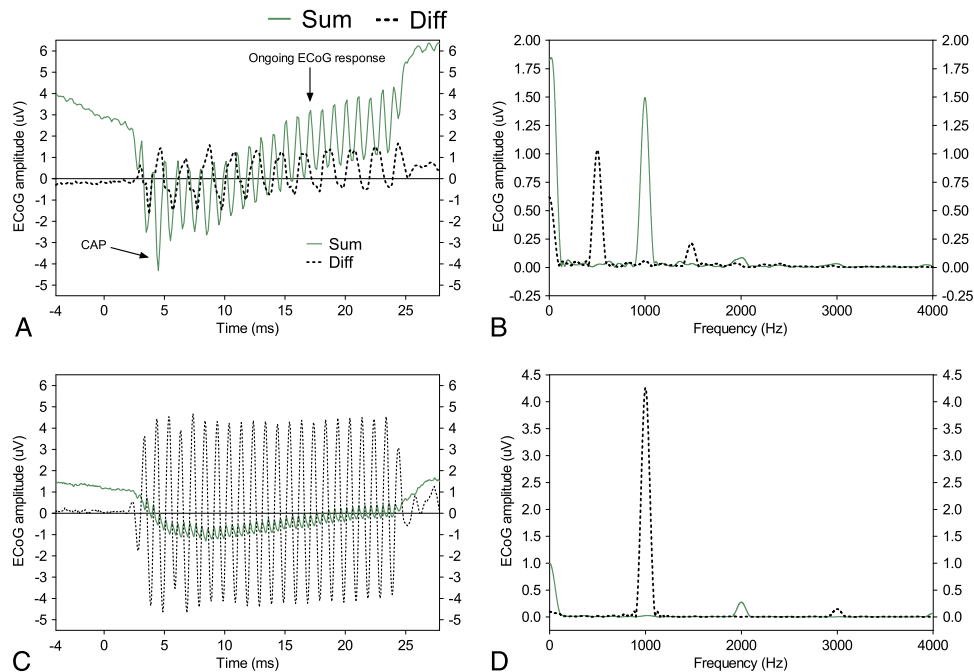


FIG. 2. Two examples for ECoG responses before insertion of the cochlear implant electrode array. Each row is a different subject. The acoustic stimuli were 500 Hz at 85 dB HL in the top row and 1000 Hz at 90 dB HL in the bottom row. The left column (A and C) is the time waveform of the ECoG responses; the right column (B and D) is the corresponding spectrum. The black line represents the difference of both ECoG responses with alternating starting phases, the green line (see online version) the average. The top row (A and B) is a case where neural contribution to the ECoG signal can be assumed. In the time waveform (A), a CAP at the beginning of the signal is visible. In the spectrum (B), a large peak is visible at the frequency of the first harmonic. In the case in the bottom row (C and D), no CAP is visible in the time waveform and the energy of the ongoing ECoG response comes almost entirely from the stimulus frequency. In the spectrum (D), only a very small peak is detectable at the frequency of the first harmonic. This suggests a dominant hair cell response.

ECoG recordings. Subject 14 showed small increases of the ECoG signal at all 3 frequencies (6.7 dB at 250 Hz, 7.8 dB at 500 Hz, and 4 dB at 1000 Hz). Subject 16 showed very small decreases at 250 and 1000 Hz and a small increase at 500 Hz (−0.3 dB at 250 Hz, 3.4 dB at 500 Hz, and −0.7 dB at 1000 Hz).

DISCUSSION

A deeper understanding of the mechanisms causing loss of residual hearing is essential to further improve hearing preservation rates after cochlear implantation. Currently, it remains unclear whether postoperative hearing loss is associated with acute cochlear trauma or whether postoperative mechanisms play a more prominent role. Therefore, the aim of this study was to monitor changes of cochlear function during surgery and to correlate these changes to postoperative hearing preservation.

To date, the primary clinical use of ECoG is in the diagnostic evaluation of Ménière's disease (22). However, recent animal studies correlating electrophysiological and histological findings in gerbils have shown that changes in the ongoing ECoG response at suprathreshold intensities are also a sensitive marker for cochlear trauma during insertion of an electrode array into the cochlea (8–12). Additionally, ECoG responses seem to be detectable in the great majority of cochlear implant re-

cipients, which further enhances their potential as a monitoring tool (13). In concordance with previously published studies, we could detect ECoG signals in 95% of cochlear implant recipients (13,15). The fact that technical problems seem to have caused the absence of ECoG signals in the only case without detectable ECoG responses in our population suggests that in subjects with residual hearing, the detectability of ECoG signals could be even higher than 95%.

The ongoing ECoG signal consists of the CM, ANN, and SP. The SP does not contribute to the energy at the signal frequency and the frequency of the first harmonic as it is a sustained depolarization for the duration of the acoustic stimulus. Therefore, it did not contribute to the amplitude of the ongoing ECoG signal as defined in our study. Consequently, we evaluated changes of the CM—a potential mainly generated by outer hair cells—and the ANN—a neural potential. Fitzpatrick et al. showed that in low frequencies, both potentials contribute to the energy at the signal frequency and at the first harmonic (23). Historically, it was assumed that the summation of 2 ECoG responses with alternating starting phases would cancel out the neural contribution to the signal and a pure CM would remain. However, this is not the case in the low frequencies. Thus, as a separation of CM and ANN was not possible, we chose the term ongoing ECoG signal in the same way as previously used in literature (15,16).

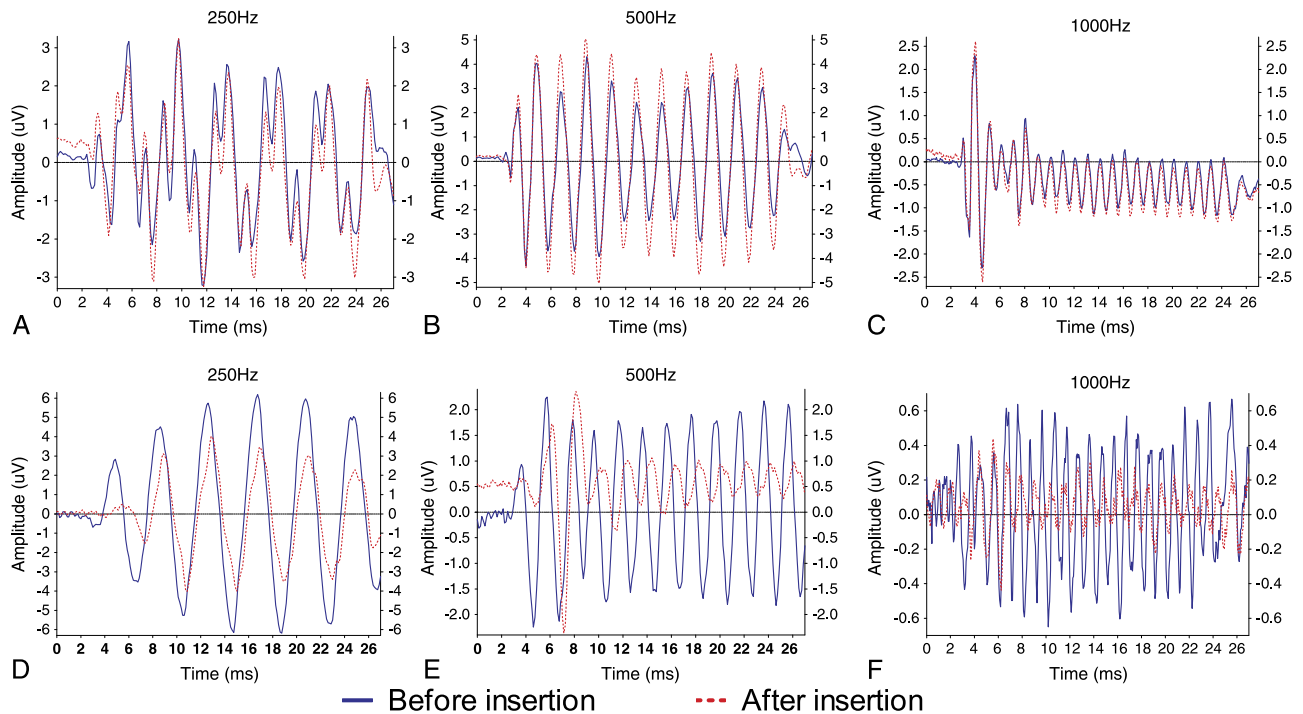


FIG. 3. Two examples for ECoG responses before and after insertion of the cochlear implant electrode array. Acoustic stimuli of 250 Hz at 80 dB HL (A and D), 500 Hz at 85 dB HL (B and E), and 1000 Hz at 90 dB HL (C and F) were used. Each row is a different subject. The left column (A and D) is the difference waveform at 250 Hz, the middle column (B and E) at 500 Hz, and the right column (C and F) at 1000 Hz. The blue line (see online version) represents the ECoG response before, the red line (see online version) the response after insertion of the cochlear implant electrode array. The case in the top row (A, B, and C) shows almost no change, whereas in the case in the bottom row, a decrease of the ECoG response amplitude after insertion of the electrode array was detectable at all 3 frequencies.

In retrospect, the signals labeled as CM in the previously published paper of Radloff et al. (5) must be considered as signals consisting of hair cell and neural responses and not pure CM. They found changes in the detection threshold of the ongoing ECoG signal (labeled as CM) in 2 of 4 subjects with deep insertions. All 4 subjects had a complete loss of residual hearing 1 week after surgery. Low frequency hearing loss in these subjects was 40 dB or larger. In our series, hearing loss around 40 dB occurred in 3 cases (subjects 10, 14, and 16). In subject 10, gross intracochlear trauma during surgery has to be assumed because of the ECoG findings. Subjects 14 and 16 showed no signs for gross intracochlear trauma in ECoG recordings despite a hearing loss of 41.7 and 38.3 dB. However, subject 14 had a rapidly progressive hearing loss on both sides after bacterial meningitis 3 months before surgery. On the contralateral side, the acoustic hearing was already completely lost at the time of surgery. Therefore, in subject 14, a further rapid progression of the hearing loss independent from cochlear implantation seems plausible. In summary, both series imply that in subjects with complete hearing loss, especially in cases with mean presurgical low frequency hearing thresholds better than 80 dB and therefore larger hearing loss, intracochlear trauma during surgery is a significant factor.

In our series, in subjects with hearing loss of 25 dB or less ($n = 14$), no signs for gross intracochlear trauma could be detected, including 2 cases (subject 12 and 15) with complete loss of residual hearing. These findings

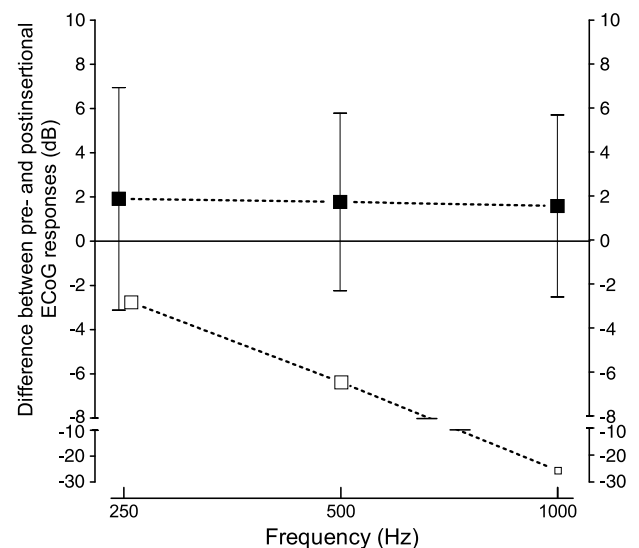


FIG. 4. The plot shows the difference in amplitude between preinsertional and postinsertional ECoG responses (y-axis) for all 3 recorded frequencies (x-axis). The black squares represent the mean amplitude change with standard deviation for all cases. The squares with no fill mark the differences in the ECoG response amplitude of subject 10. This pattern with decreases in ECoG responses for all recorded frequencies was associated with a complete hearing loss 4 weeks after surgery.

suggest postoperative changes or minor cochlear trauma to more basal cochlear regions—not detectable by ECoG recordings in low frequencies—as underlying mechanisms of threshold shifts in such cases.

An interesting finding is that in the series of Radeloff et al. (5) and in our series, all subjects with detectable threshold changes or amplitude changes in ECoG responses had a complete loss of residual hearing. This implies that hearing preservation in a case with detected cochlear trauma by use of extracochlear ECoG recordings in low frequencies seems unlikely. Especially in candidates for electric-acoustic stimulation in which a limited insertion depth is intended, such a finding could indicate the need for full insertion to obtain more benefit from electrical stimulation alone. In contrast, especially in cases with limited residual hearing in which a hearing loss of 25 dB or less leads to complete loss of residual hearing, the absence of detectable trauma in low frequency ECoG recordings does not exclude complete hearing loss. Therefore, changes in ECoG recordings as described in the series of Radeloff et al. and in our series seem to have a high specificity for complete loss of residual hearing but a limited sensitivity.

As outlined previously, in subject 14, a progression of hearing loss independent from cochlear implantation seems plausible. Apart from that, only in one case signs for a progression of hearing loss independent from cochlear implantation occurred. In subject 19, a change of greater than 10 dB in low frequency hearing thresholds was detectable on the contralateral side. In all other subjects—due to pure tone audiogram findings on the contralateral side and medical history—a rapid loss of residual hearing on the operated side independent from cochlear implantation seems unlikely.

An unexpected finding was that increases in ECoG signals were present in multiple subjects. An increase exceeding 5.1 dB (mean plus 1.96 standard deviations in repeated ECoG recordings under unchanged conditions) occurred in 6 cases at 1 or 2 frequencies. Small increases can be explained with variability in recordings. For larger increases, different explanations are possible: 1) contact of the recording electrode with the perilymph due to placement near the round window, although the round window was sealed with soft tissue before conducting the postinsertional recordings, or 2) pressure changes in the scala tympani and scala vestibuli due to insertion of the cochlear implant electrode. The second explanation arises from animal studies demonstrating a close relation between the amplitude of CM and the pressure difference between scala tympani and vestibuli (24). Whereas the second explanation would represent a process inside the cochlea as a result of the cochlear implantation, the first explanation could represent limitations of the recording technique. A possible trauma causing a decrease of the ECoG signal could be concealed by an increase of the signal caused by changes of the recording conditions. Recordings from more distant extracochlear sites could be a solution to this problem. However, signal quality and signal-to-noise ratio could limit the value of more distant recording sites. In our own experience, recordings with a

recording electrode placed on the horizontal semicircular canal showed no clear ECoG signal.

CONCLUSION

The described technique for extracochlear ECoG recordings is a reliable tool to assess cochlear function during cochlear implantation. A decrease of ECoG signals at suprathreshold intensities in multiple frequencies indicates gross cochlear trauma and seems to be predictive of complete loss of residual acoustic hearing. In cases with moderate threshold shifts of 25 dB or less, ECoG recordings in low frequencies showed no changes. This could indicate that postoperative mechanisms or minor cochlear trauma to more basal cochlear regions are responsible for loss of residual hearing. Further studies are required to fully elucidate the role that intraoperative ECoG recordings may have in monitoring cochlear trauma and residual hearing during cochlear implantation.

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